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Anticipatory and reactive response to falls: Muscle synergy activation of forearm muscles.
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Abstract

We investigated the surface electromyogram response of six forearm muscles to falls onto the outstretched hand. The extensor carpi radialis longus, extensor carpi radialis brevis, extensor carpi ulnaris, abductor pollicis longus, flexor carpi radialis and flexor carpi ulnaris muscles were sampled from eight volunteers who underwent ten self-initiated falls. All muscles initiated prior to impact. Co-contraction is the most obvious surface electromyogram feature. The predominant response is in the radial deviators. The surface electromyogram timing, we recorded would appear to be a complex anticipatory response to falling modified by the effect on the forearm muscles following impact. The mitigation of the force of impact is probably more importantly through shoulder abduction and extension and elbow flexion rather than action of the forearm muscles.

Introduction

The parachute reaction of upper limb extension and slight abduction with extension of the wrists, thumb and digits in response to quick approach to a visual surface first appears in infants from 4 months of age and is well developed by 9 months. It is considered a postural response under visual and vestibular control.[1] Protective arm movements detected by EMG activity in deltoid are seen less than 100ms after perturbation in platform translation studies.[2]

In a fall on to the outstretched hands, protection from injury can be divided into three stages; detection of imbalance, movement of the hands in to the landing position and energy absorption during impact.[3] Muscle preactivation can alter the viscoelastic property of muscle to quickly compensate for impact.[4] The force of impact can also be attenuated by muscle, which can passively deform and eccentrically contract.[5] Modulations of joint angles (elbow flexion and shoulder extension/abduction) also act to dissipate force. Muscle contraction however increases stiffness in the segment and transmits greater force proximally up the limb. Burkhart TA and Andrews DM[6] suggested more work was needed to determine the relationship between stiffness and stability, and which forearm muscles were most involved in this process.

The sEMG activity in triceps has been studied in falls on to the outstretched hands and both preexisting activity and the spinal stretch reflex are thought to be important to triceps activity after impact.[4] There have however been limited sampling of forearm muscles in fall studies describing the sEMG response in proximal limb muscles.[5-7]

Safely landing from jumping or falling requires accurate assessment of time to contact to allow for appropriate muscle preactivation.[6,8-9] Study into the EMG activity in leg muscles with landing onto the feet provide insight into muscle preactivation.[10] By conventional muscle activation is divided into pre and post landing, but in reality it is a continuum extending from initiation to completion of the fall.[10] Because ground reaction forces can rise within 80ms, reflexes alone would not act quickly enough to adequately position the limbs or absorb the forces of impact.[3-4,11] Increased muscle preactivation is required to prepare for impact and can be achieved by varying the onset of activation or the rate at which the activity increases. The reflex response to impact can influence the EMG activity but does not initiate or control it.

The preparatory or anticipatory set of muscle activity in a reflex action has been studied in detail in the physiology literature.[8,12-13] A complex action (such as catching or throwing) can be divided into a chronological set or timing based and a topographical set or task based. Visual input has been shown to be able to modulate the mid and long latency stretch reflex if the load on the muscle is anticipated.[14]

In 1997, Petrie et al.[15] first reported the identification of mechano-receptors in the palmar wrist ligaments. However, the exact role of mechano-receptors in stability and function of the wrist remains unclear, although the inference is that proprioceptive afferents are able to contribute to wrist stability by allowing central modulation of wrist muscle activity (long loop reflexes 70-90ms delay or spinal type stretch reflexes 20 -30 ms delay).[16-18]

Since there are no tendons directly attaching to the carpal bones, their position is dependent on the compressive forces generated by the muscles acting across the joint and the shape of the articular contours.[19] From the 1980's investigators postulated a role specific muscles in influencing the position of individual carpal bones,[20] although the it was not till later that the mechanism were suggested.

Salva-Coll et al. [21-22] investigated the role of the flexor carpi radialis (FCR) on stabilizing the scaphoid and concluded that FCR acted to supinate the distal carpal row therefore supinating the scaphoid via the scapho-trapezial joint. Scaphoid supination closes the dorsal scapho-lunate interval therefore detensioning and protecting the scapholunate ligament (SLL). They introduced the concept of scapho-lunate "friendly" distal carpal row supinators and "unfriendly" distal carpal row pronators such as extensor carpi ulnaris (ECU).

Hagert et al.[17] described wrist ligamento-muscular reactions using electrical stimulation of the scapholunate ligament and recording sEMG over the forearm muscles. A direct monophasic reaction was identified at 20ms post stimulation in the antagonist muscles, followed by agonist activity after 20–60ms. Co-contraction was noted with peaks after 150ms. The response depended on the position of the wrist at the time of SLL stimulation that is in extension the flexor muscles were initially activated and in flexion it was the extensor muscles that demonstrated an initial response.

The effect of excessive extension, ulnar deviation and supination leads to sequential failure of the carpal ligaments around the lunate.[23] Where uncontrolled hyperextension of the wrist occurs the scaphoid extends more than lunate leading to tearing of scapholunate ligament. Where hyperextension does not occur the impact may lead to wrist fracture.[11] In ulnar deviation the proximal scaphoid moves radially away from the lunate; this is seen in normal wrists with ulnar deviation. If the long Radio Lunate ligament limits lunate ulnar deviation, as it seems to in CT modeling studies,[24]-[25] then further ulnar deviation of the scaphoid would stress the SL ligament. Radiocarpal pronation-supination is fairly restricted in most individuals. The forced pronation of the forearm about a fixed hand leads to carpal supination. If the lunate supinates less than the scaphoid then the dorsal SL interval narrows. This theoretically protects the dorsal band of the scapholunate ligament, but stresses the volar band of the ligament.

With the wrist in extension and the forearm in pronation the action of the forearm muscles is slightly different to when the wrist is in the neutral position. The extensor carpi radialis longus (ECRL) extends, radially deviates and probably pronates the wrist when it is in extension. The extensor carpi radialis brevis (ECRB) extends the wrist and is a weak radial deviator and minimal pronator of the carpus. The ECU is a weak extensor but strong ulnar deviator with the forearm in pronation. It causes carpal supination. The abductor pollicis longus (APL) is a flexor and strong radial deviator. APL causes carpal pronation. The FCR is a flexor and radial deviator. It causes carpal pronation. The flexor carpi ulnaris (FCU) is the strongest wrist flexor and an ulnar deviator of the carpus. FCU probably supinates the extended carpus. In the fall on to the extended wrist extension would be opposed by FCR, FCU and APL, ulnar deviation would be opposed by ECRL, APL and FCR. Carpal supination would be opposed by FCU and ECU.

There is an increasing interest in proprioception in the wrist but incomplete understanding of the place of protective reflexes in mitigating injury in forces applied to the wrist. There have been only limited studies of EMG activity in the forearm in falls and these have looked at grouped flexor and extensor responses[4] or single muscles such as FCR[6] or ECU.[5] With this in mind we undertook a pilot study to investigate the sEMG in six forearm muscles in response to impact in self-initiated falls. If there were a protective reflex we would expect that it would act to oppose excessive extension, ulnar deviation and perhaps supination.

Materials and Methods

Participants

Eight participants were recruited from first year medical students. There were seven males and one female. Average age was 23 .6 years (range 21 – 46 years). Informed consent was obtained from the participants and the experiment was carried out under the guidelines of the Human Research Ethics Committee of the Queensland University of Technology.

Procedure

Surface EMG was recorded whilst the participants performed four tasks. Flexion extension motion, rapid wrist extension, passive wrist extension and self-initiated falls. Slow rhythmic flexion extension motion (one cycle per two seconds) was performed against gravity with the forearm pronated and the forearm supported in a horizontal position. Rapid voluntary wrist extension was performed from the same forearm position with the wrist starting in the neutral position. A hand surgeon (GC) performed rapid passive wrist extension on each participant with the forearm stabilized in the pronated position starting from the wrist in neutral. The participants each underwent ten self-initiated forward falls from a kneeling position landing on a rubber mat. The arms were by the side at the commencement of the fall. The participants were instructed to land on the palm of their hands and not make first contact with the fingertips.

Surface electromyogram

The surface electromyogram (sEMG) activities in ECRL, ECRB, ECU, APL, FCR, and FCU were recorded in the right forearm. Bipolar sEMG recordings were made using a wireless system (ZeroWire, Noraxon, Arizona USA). The skin was prepared by shaving and wiping with an alcoholic swab. Two surface electrodes (20mm diameter) 25mm apart were placed on the skin over the surface markings for the belly of the muscle to be studied.[26] The sEMG was sampled at 2,000 Hz. A Butterworth type filter with a high pass of 5Hz and low pass of 500 Hz was used.

An event pressure switch (Motion Lab Systems MA-153) was placed on the thenar and hypothenar eminences. This allowed us to identify the timing of impact. The event switch responds to 200g pressure and timing of impact was taken as the first deflection detected from the switch.

Analysis

The surface EMG data was then exported and analyzed using the software MyoResearch XP (Noraxon). The sEMG signal was full wave rectified and smoothed with a low pass filter with a time constant of 10ms, and the profile plotted. A template of the smoothed full wave rectified trace was defined based on five reproducible points at discrete changes in slope of the sEMG trace. The sEMG was segmented into initiation from baseline, preparatory activity, pre peak slope, peak amplitude, and return to baseline signal (Figure 1 and 2). Each sEMG was then characterized by the pre peak slope into those falls with an increase, decrease or no change in gradient occurring prior to peak amplitude. The points in time corresponding to the template points were then identified on the sEMG data for each muscle along with the time of contact. Surface EMG electrical profiles were then created for each muscle for individual participants by averaging each of the time periods. An overall profile was created for each of the six muscles by averaging the muscle profiles of the participants.

Results

Surface electromyogram

The initial activity is seen in ECRB as the wrist is extended following the initiation of the fall. The overall response to impact of the forearm muscles studied is one of co-contraction (figure 3). Generally the extensors are more active and initiate and peak before the flexors. Three patterns were observed. The commonest was that the radial wrist extensors would peak first (six out of eight participants); in one participant the FCR peaked with the extensors. In two participants all the extensors activated. The average time period spanning the peaks of all six muscles was from 26 to 89ms post impact.

Initiation

The initiation of sEMG activity above baseline was detected between 240ms and 80ms prior to impact. ECRB appeared to be the main muscle post impact responding faster and to a higher peak than the other muscles. It was the first muscle to initiate in all eight participants. In four participants ECRL initiated at the same time. In two participants ECRB and ECU were the first muscles to activate and in one of these APL also activated simultaneously. All the extensors had activated within 20ms of ECRB. FCR and FCU activated on average 35ms after ECRB. All muscles in all participants recorded an initial deflection from baseline prior to impact being detected by the pressure sensor.

Peak amplitude

The peak in extensor sEMG activity occurred on average 26ms after impact for ECRL, 40.5ms after impact for ECRB and at 44.5ms for ECU. There was a similar range of 20 to 120ms for all three muscles. In the flexor muscles, FCU consistently peaked first averaging 75.2ms from impact. APL and FCR peaked at a similar time (average 87.5 and 89.2ms respectively). The range was from 40ms to 140ms from impact.

Double peaks were seen in the recordings of all muscles, most commonly in ECRB (32% of falls). It was least commonly seen in the sEMG of FCR (10%) and FCU (12%). Where a double peak occurred in the sEMG trace one of the peaks would always coincide with the peak from another muscle. The commonest of these were ECRB and APL or ECRB, APL and FCR. Without synchronous kinematic information from wrist position it is not possible to determine the role or effect of the amplitude double peak.

Associated muscle responses

The response of the muscles studied to impact was variable between participants and the falls of each participant. Two by two tables were created to determine associations between positive and negative responses in muscle pairs. Significant associations were identified using Fischer's exact test ($p>0.05$). Strong associations were seen between APL and FCR, ECRB and ECRL, FCU and FCR, and FCU and ECU. When APL exhibited a positive response FCR was positive in 94%. When APL was negative FCR was negative in 88%. When ECRB was positive ECRL was always positive. When FCU was positive FCR was always positive. When FCU was negative, ECU was negative in 82%. No associations were identified for antagonist pairs (eg FCR/ECU, radial wrist extensors/FCU).

The normalized amplitudes for positive and negative responses in the muscles were similar for all muscles varying between 0.5 – 0.68 for the positive response and 0.65 – 0.75 for the negative response (table 1). The pre peak activation occurs earlier in the negative response except in FCR (table 2). FCR was the only muscle in which the negative response occurred from a pre peak after impact.

In general the negative response occurs earlier from higher normalized amplitude than the positive response.

Timing

The peak amplitude occurred after impact for all muscles. The peak amplitudes occurred earlier in the negative responses of ECRB, ECU, APL, and FCU (table 2). In FCR the peak amplitude occurred earlier in the positive response. In ECRL there was no difference in the timing of the peak amplitude. The largest difference between positive and negative response was in ECU (positive 57ms, negative 2ms). The normalized amplitudes for each of the studied muscles for positive and negative responses is plotted against time in figures 4-9.

Comparison with wrist motion sEMG

The sEMG recordings made during the simulated falls for each participant were compared to recordings made during active flexion extension motion, active rapid extension and passive rapid extension. In slow flexion extension (1 cycle per 2 seconds) the amplitudes are lower across all muscles and the slopes of the traces are less steep than those seen in the fall recordings. The lowest amplitudes are seen in rapid passive extension. The average extensor amplitudes reflect approximately 10-20% (ECRL: 13.8%, ECRB: 11.5%, and ECU: 17.6%) of the extensor amplitudes recorded in falls and the flexor values are lower (APL: 5.6%, FCR 5.8%, and FCU 5.6%). The six muscles all initiate simultaneously. The flexors peak approximately 20ms before the extensors, which is the reverse of what was observed in the simulated falls.

In rapid active extension of the wrist, the sEMG recording of the extensor muscles and APL demonstrated steeper slopes than in the simulated falls. The response recorded in the FCR and FCU was more similar to that seen in falls. Overall the muscle response was more synchronous in rapid active extension than in falls with all peaks occurring within 10-20ms of the initial peak in ECRB and APL.

The extensor and APL amplitudes tended to be lower in falls than in rapid active extension (ECRL: 78%, ECRB: 84%, ECU: 71%, and APL 68%). The amplitude in the FCR and FCU tended to be lower (average FCR 33.5%, and FCU 38%) although one participant recorded a value representing 150% of the FCU amplitude recorded in the falls.

Video

Review of the video confirmed the sequence of joint motion occurring with fall onto the outstretched wrist. After impact the wrist extends and ulnar deviates and the carpus supinates relative to the forearm as the forearm pronates around the fixed hand. The elbow flexes and the shoulder extends with a variable amount of abduction. Between participants the time from initiation of the fall to impact ranged from 840ms to 960ms. The time from impact to loss of forward momentum varied from 200ms to 320ms. There is a brief period of wrist extension that occurs immediately after impact coinciding with rapid elbow flexion.

Discussion

We studied the sEMG recording from six forearm muscles during self-initiated falls onto the outstretched hands from a kneeling position. A pressure sensor was used to determine time of impact. Co-contraction is the most obvious feature in the recorded falls with all muscles peaking within approximately 70ms of each other. In the extended wrist position co-contraction of the radial and ulnar deviators or strong contraction of the radial deviators (ECRL, ECRB, APL, and FCR) against a wrist which is being ulnar deviated in a fall would act to compress the dorsal Scapholunate interval and protect the dorsal band of the scapholunate ligament. Co-contraction also possibly limits relative carpal bone motion in the closed pack position of extension protecting the intercarpal ligaments. Co-contraction is similarly the major feature in the leg muscles in landing on the feet from a height[9] and the forearm muscles in catching a ball.[8]

We expected to see greater activity in the flexors, acting to resist forced extension of the wrist. There was neither the amplitude nor the rapid excitation that one would expect to observe if there was a protective reflex present. The initiation of flexor activity occurred prior to the detected impact in all recorded falls and the peak in amplitude 80-90ms after impact. It is hypothesized that the stretch reflex can be preset by central anticipatory mechanisms gating a pattern of co excitation, rather than the typical stretch response in antagonist muscles.[8] A stretch reflex would be expected to be detected 20-30ms after impact if this was to be present. FCR was the only muscle in which the pre peak activation was consistently initiated post impact, and peak activity in all muscles subsided in under 100ms post impact. The activity in FCR bears further investigation since it has been suggested as acting to protect the SLL.[21]

ECU is a carpal pronator and as such stresses the dorsal SLL. We did not observe a significant trend of ECU decreased activity with impact. A negative response in ECU was observed in 47% of falls.

Ulnar deviation of the carpus uncovers the scaphoid perhaps making it more prone to injury and also allowing the scaphoid to hinge more on the dorsal lip of the radius placing more force proximally through the attachment to the lunate. The relative radial extension moment produced by the wrist motors may protect the carpus by resisting carpal ulnar deviation and holding the scaphoid more in the scaphoid fossa of the radius.

Either the amplitude of the peak value or the latency between the onset of the preactivation and the peak determines the positive or negative response of the pre peak activation gradient. The greater the amplitude difference and the shorter the latency cause a more positive response. It is tempting to surmise that there is a mechanism by which the EMG activation is increased or decreased in preparation for impact, but elucidation of this requires further study.

There are a number of limitations in a study of this nature. We used self-initiated low velocity falls to reduce the risk of injury but this means that the visual and vestibular reflexes can contribute to the response. Anticipation allows loading of a preset motor response which can be activated at very short latencies (<170ms) .The low energy fall does not hyperextend the wrist as much as would occur in a more violent fall.

From figures four to nine we can see initiation and rapid ramping up of the normalized sEMG occurring prior to ground contact. Fifty to 90% of the peak amplitude recorded has occurred by impact. The pre peak phase in both positive and negative responses occur prior to impact in all muscles except FCR. The extensors peaked 20-120ms after contact (average latency ECRL 26ms, ECRB 40.5ms, and ECU 44.5ms) and the flexors peaked 40-140ms after contact (average latency FCU 75.2ms, APL 87.5ms, and FCR 89.2ms). This latency is within the 30-35ms one would expect for a simple stretch reflex and the longer loop reflexes of 50-

70ms.The later peaks occurring after 100ms are in the time frame for the late spinal reflex phase and the sub cortical motor programs loaded for falls. The sEMG timing we recorded would appear to be a complex anticipatory response to falling modified by the effect on the forearm muscles following impact. The extension of the wrist is initiated and occurs prior to impact. The muscle activity is then modified by the impact presumably to adjust the position of the wrist. The flexor muscles initiate prior to impact in a similar fashion. Pre-activity may prepare the muscle for more rapid or stronger contraction but it seems unlikely based on the amplitudes and slope of the sEMG traces that the wrist flexors act to mitigate the force of the fall. If there is a protective reflex then based on muscle activity recorded in this study it appears it is the radial deviators that are active to oppose ulnar deviation rather than the flexors opposing forced hyperextension. The absorption of the force of impact is probably more importantly through shoulder abduction and extension and elbow flexion than action of the forearm muscles. Further studies are planned to determine the contribution of anticipatory, vestibular, visual and impact to the sEMG and kinematics of the wrist in falls onto the hands. A better understanding of the muscle activity and the kinematics of falls onto the outstretched hands will allow further insight into mechanism of injury and strategies for injury prevention.

References

1. Wenzel D. The development of the parachute reaction-a visuo-vestibular response. Neuropadiatrie. 1978;9(4):351–9.
2. McIlroy WE, Maki BE. Early activation of arm muscles follows external perturbation of upright stance. Neurosci Lett. 1995;184(3):177–80.
3. Robinovitch SN, Normandin SC, Stotz P, Maurer JD. Time requirement for young and elderly women to move into a position for breaking a fall with outstretched hands. J Gerontol A Biol Sci Med Sci. 2005;60(12):1553–7.
4. Dietz V, Noth J, Schmidtbleicher D. Interaction between pre-activity and stretch reflex in human triceps brachii during landing from forward falls. J Physiol. 1981;50:113–25.
5. Burkhart T a., Andrews DM. Activation level of extensor carpi ulnaris affects wrist and elbow acceleration responses following simulated forward falls. J Electromyogr Kinesiol. 2010;20(6):1203–10.
6. Burkhart TA, Andrews DM. Kinematics. Kinetics and muscle activation patterns of the upper extremity during simulated forward falls. J Electromyogr Kinesiol. 2013;23(3):688–95.
7. Corbeil P, Bloem BR, van Meel M, Maki BE. Arm reactions evoked by the initial exposure to a small balance perturbation: A pilot study. Gait Posture. 2013;37(2):300–3.

8. Lacquaniti F, Maioli C. The role of preparation in tuning anticipatory and reflex responses during catching. J Neurosci. 1989;9(1):134–48.

9. Santello M, McDonagh MJ. The control of timing and amplitude of EMG activity in landing movements in humans. Exp Physiol. 1998;83(6):857–74.

10. Santello M. Review of motor control mechanisms underlying impact absorption from falls. Gait Posture. 2005;21(1):85–94.

11. Troy KL, Grabiner MD. Asymmetrical ground impact of the hands after a trip-induced fall: experimental kinematics and kinetics. Clin Biomech. 2008;22(10):1088–95.

12. Carlsen AN, Maslovat D, Franks IM. Clinical neurophysiology preparation for voluntary movement in healthy and clinical populations : Evidence from startle. Clin Neurophysiol. 2012;123(1):21–33.

13. Stevenson AJT, Chiu C, Maslovat D, et al. Cortical involvement in the startreact effect. Neuroscience. 2014;269:21–34.

14. Yamamoto C, Ohtsuki T. Modulation of stretch reflex by anticipation of the stimulus through visual information. Exp Brain Res. 1989;77(1):12–22.

15. Petrie S, Collins J, Solomonow M, Wink C, Chuinard R. Mechanoreceptors in the palmar wrist ligaments. J Bone Joint Surg Br. 1997;79(3):494–6.

16. Hagert E. Proprioception of the wrist joint: a review of current concepts and possible implications on the rehabilitation of the wrist. J Hand Ther. 23(1):2–16; quiz 17.

17. Hagert E, Persson JKE, Werner M, Ljung B-O. Evidence of wrist proprioceptive reflexes elicited after stimulation of the scapholunate interosseous ligament. J Hand Surg Am. 2009;34(4):642–51.

18. Vekris MD, Mataliotakis GI, Beris AE. The scapholunate interosseous ligament afferent proprioceptive pathway: a human in vivo experimental study. J Hand Surg Am. 2011;36(1):37–46.

19. Kijima Y, Viegas SF. Wrist anatomy and biomechanics. J Hand Surg Am. 2009;34(8):1555–63.

20. Kauer JM. Functional anatomy of the wrist. Clin Orthop Relat Res. 1980;(149):9–20.

21. Salva-Coll G, Garcia-Elias M, Leon-Lopez M, Llusa-Perez M, Rodriguez-Baeza A. Effects of forearm muscles on carpal stability. J Hand Surg Eu. 2011;36(7):553–9.

22. Salva-Coll G, Garcia-Elias, M, Leon-Lopez, M-T, Llusa-Perez M. Role of the extensor carpi ulnaris and its sheath on dynamic carpal stability. J Hand Surg Eu. 2012;37(6):544-8.

23. Mayfield J, Johnson R, Kilcoyne R. Carpal dislocations: pathomechanics & progressive perilunar instability. J Hand Surg Am. 1980;5(3):226–41.

24. Tan J, Chen J, Tang JB. In vivo length changes of wrist ligaments at full wrist extension. J Hand Surg Eu. 2014;39(4):384–90.

25. Xu J, Tang JB. In vivo length changes of selected carpal ligaments during wrist radioulnar deviation. J Hand Surg Am. 2009;34(3):401–8.

26. DiDomenico A, Nussbaum M a. Estimation of forces exerted by the fingers using standardised surface electromyography from the forearm. Ergonomics. 2008;51(6):858–71.

Tables

Table 1. Average sEMG activity normalized to peak amplitude for positive and negative responses for each muscle. SD in shown brackets. Note higher pre peak values for the negative responses.

	Initiation	Preparatory	Pre peak	Peak	Post
ECRL +ve	0.07(0.01)	0.18(0.1)	0.50(0.08)	1.00	0.25(0.15)
ECRL -ve	0.08(0.04)	0.34(0.13)	0.73(0.12)	1.00	0.31(0.12)
ERCB +ve	0.10(0.05)	0.38(0.14)	0.68(0.08)	1.00	0.26(0.14)
ERCB -ve	0.12(0.07)	0.39(0.11)	0.70(0.1)	1.00	0.31(0.19)
ECU +ve	0.05(0.01)	0.27(0.12)	0.54(0.09)	1.00	0.28(0.14)
ECU -ve	0.11(0.04)	0.30(0.11)	0.65(0.11)	1.00	0.27(0.13)
APL +ve	0.09(0.04)	0.23(0.06)	0.53(0.16)	1.00	0.32(0.2)
APL -ve	0.06(0.04)	0.20(0.1)	0.68(0.22)	1.00	0.22(0.13)
FCR +ve	0.10(0.05)	0.34(0.11)	0.66(0.13)	1.00	0.34(0.14)
FCR -ve	0.08(0.05)	0.35(0.08)	0.75(0.1)	1.00	0.29(0.16)
FCU +ve	0.05(0.02)	0.22(0.08)	0.56(0.11)	1.00	0.21(0.11)
FCU -ve	0.08(0.04)	0.23(0.1)	0.74(0.14)	1.00	0.27(0.1)

Table 2. Average time in milli-seconds relative to impact at 0 seconds. Negative values represent pre impact. SD shown in brackets.

	Initiation	Preparatory	Pre peak	Peak	Post peak
ECRL +ve	-175(91)	-78(22)	5(7)	45(14)	187(34)
ECRL -ve	-280(89)	-93(26)	-11(14)	42(16)	216(28)
ERCB +ve	-240(95)	-137(32)	-29(8)	31(13)	214(60)
ERCB -ve	-395(67)	-202(40)	-77(11)	11(7)	246(53)
ECU +ve	-337(108)	-90(21)	7(39)	57(18)	436(102)
ECU -ve	-372(103)	-142(43)	-70(17)	29(8)	227(106)
APL +ve	-281(116)	-104(26)	20(13)	76(17)	262(96)
APL -ve	-393(106)	-80(12)	-24(14)	25(9)	458(100)
FCR +ve	-232(111)	-70(14)	13(3)	58(28)	194(72)
FCR -ve	-171(68)	-60(25)	26(4)	83(32)	266(98)
FCU +ve	-104(64)	-62(15)	20(7)	62(16)	149(85)
FCU -ve	-233(112)	-109(24)	-10(9)	46(12	249(77)

Figures

Figure 1. Template for positive sEMG trace. Arbitrary scale for time x axis and amplitude y axis. Each full wave rectified, smoothed sEMG trace was interpreted based on the template identifying the points of significant change in slope. The slope of the pre peak activity is steeper than the preparatory phase.

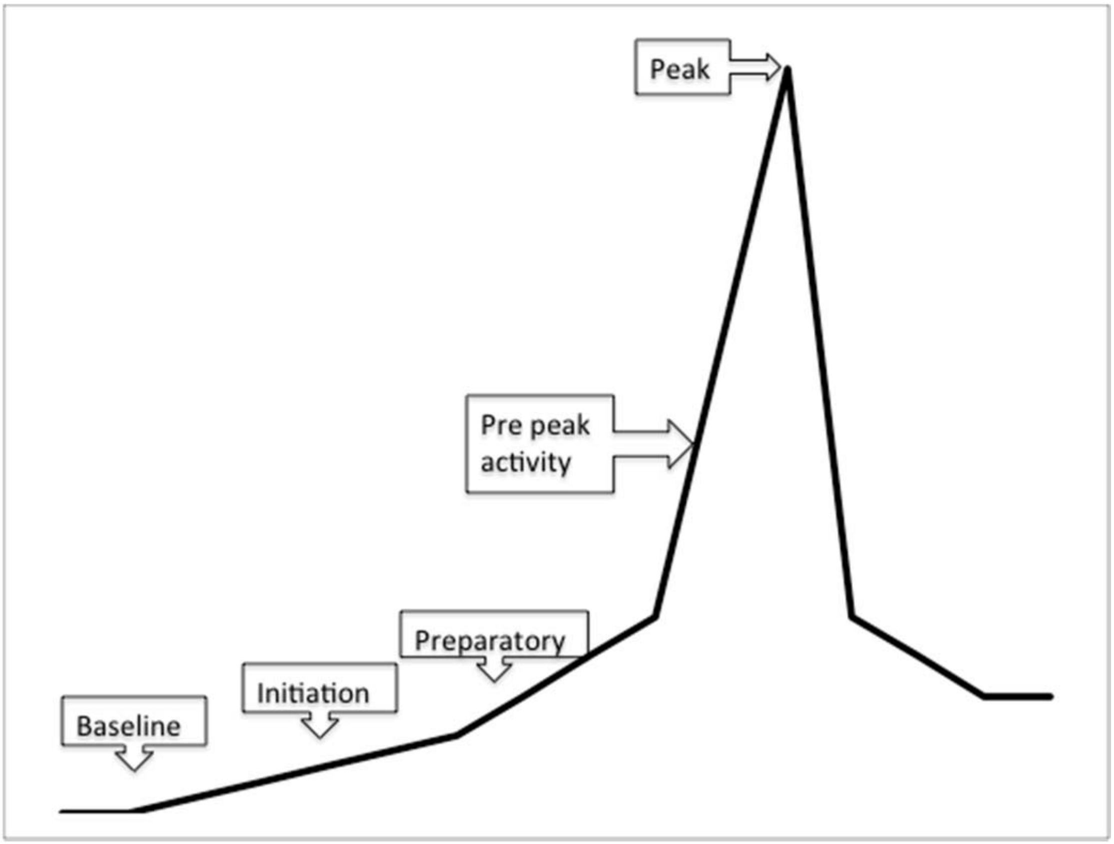

Figure 2. Template for negative sEMG trace. Arbitrary scale for time x axis and amplitude y axis. Each full wave rectified, smoothed sEMG trace was interpreted based on the template identifying the points of significant change in slope. The slope of the pre peak activity is less steep than the preparatory phase.

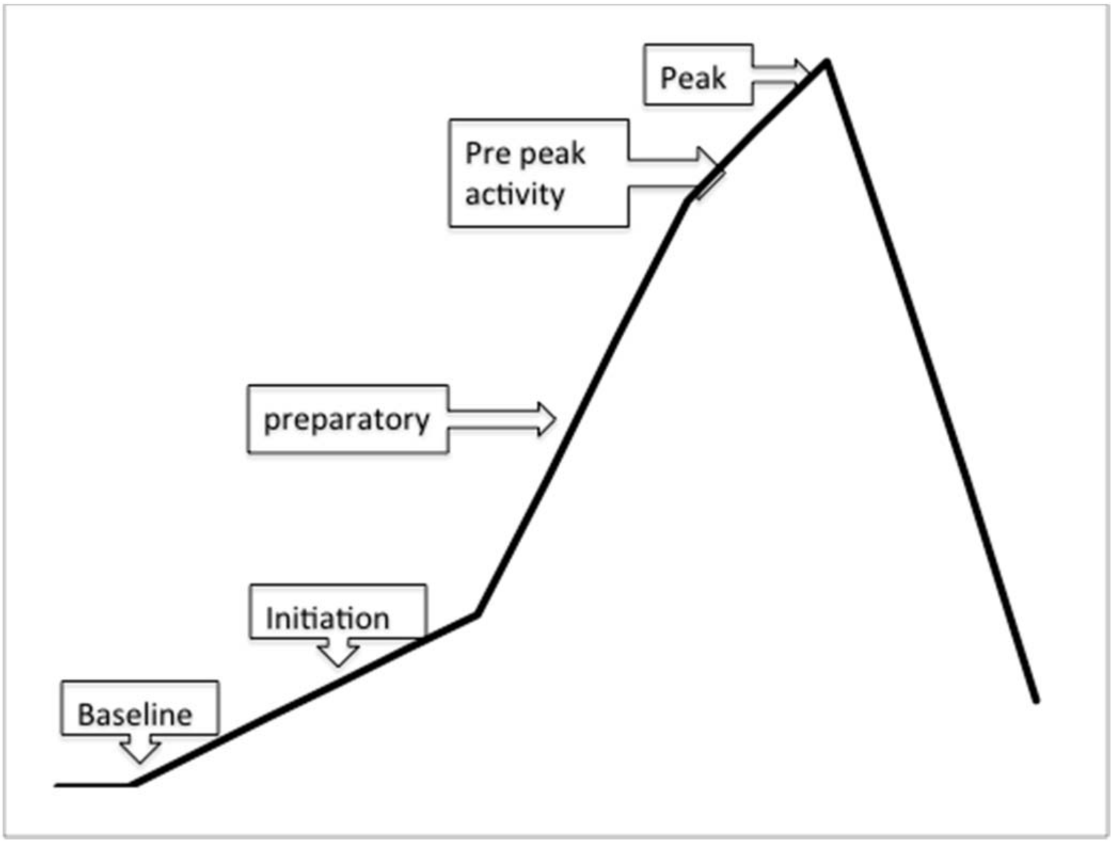

Figure 3. Example of raw sEMG log, and data collection interface (Myores). Vertical line represents point of maximum ground reaction force. Contact occurs approximately 60-80 milliseconds prior to this.

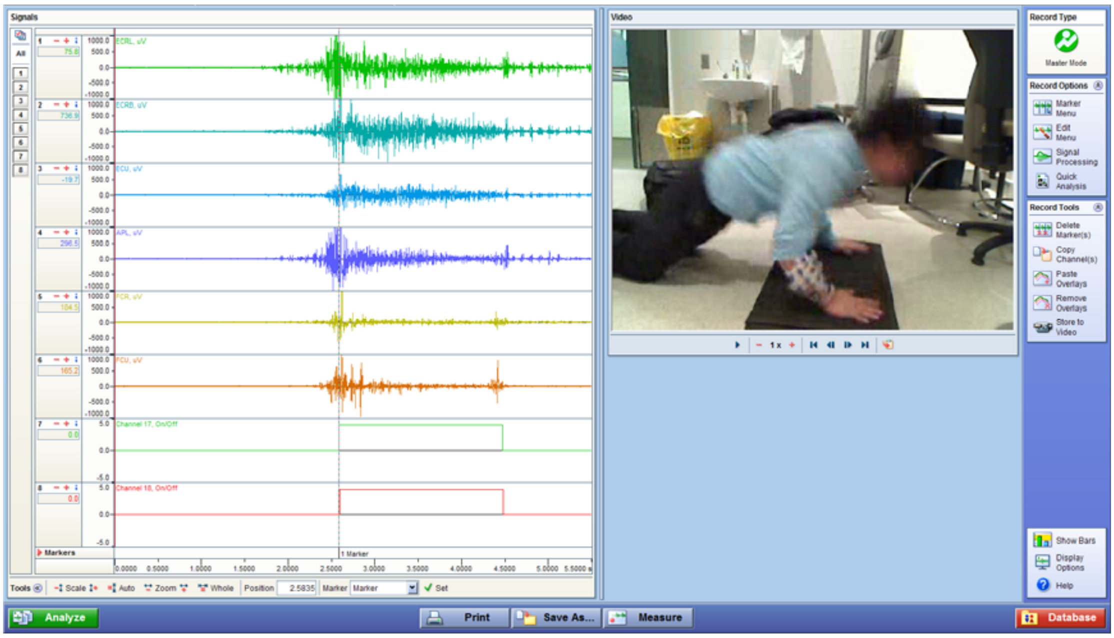

Figure 4. ECRL average sEMG amplitude normalized to peak plotted against time in milliseconds. Impact is plotted at O seconds. Note the negative response occurring earlier from higher amplitude.

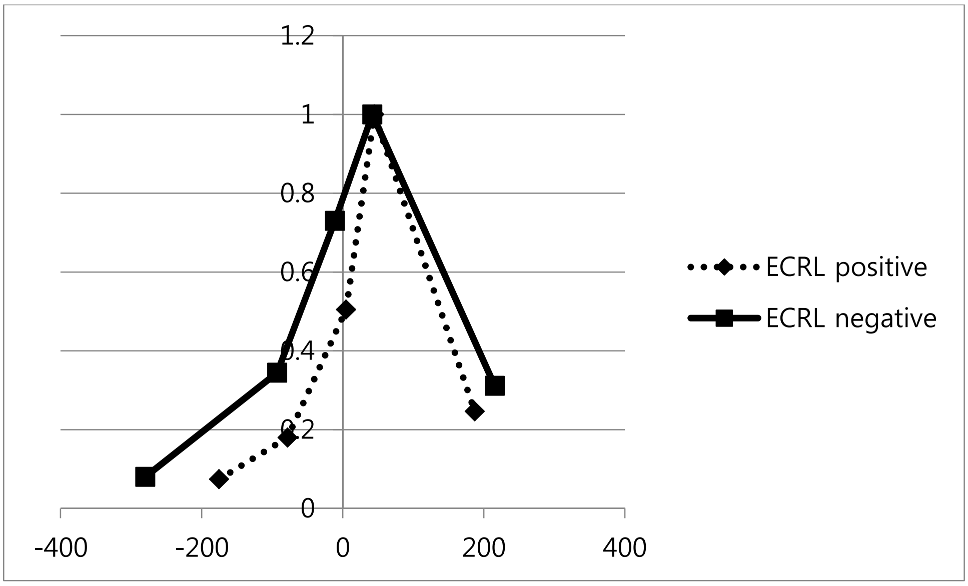

Figure 5. ECRB average sEMG amplitude normalized to peak plotted against time in milliseconds. Impact is plotted at O seconds. Note the negative response occurring earlier from higher amplitude.

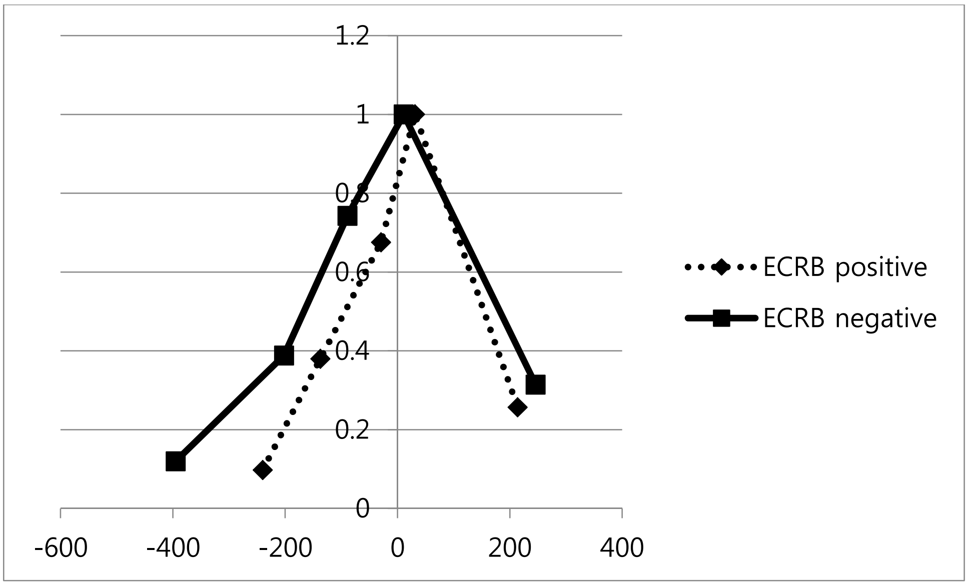

Figure 6. ECU average sEMG amplitude normalized to peak plotted against time in milliseconds. Impact is plotted at O seconds. Note the negative response occurring earlier from higher amplitude and earlier peak.

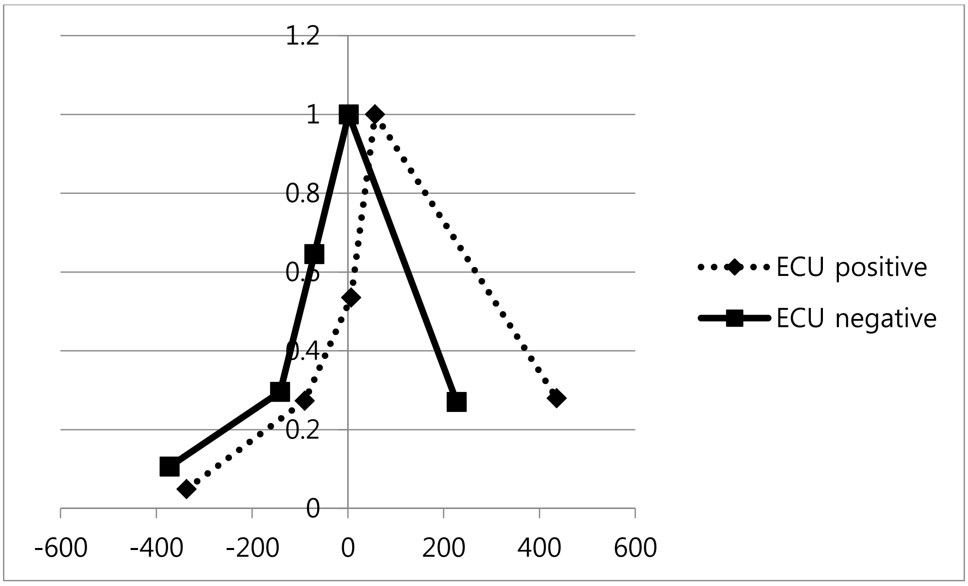

Figure 7. APL average sEMG amplitude normalized to peak plotted against time in milliseconds. Impact is plotted at O seconds. Note the negative response occurring later from lower amplitude and higher peak.

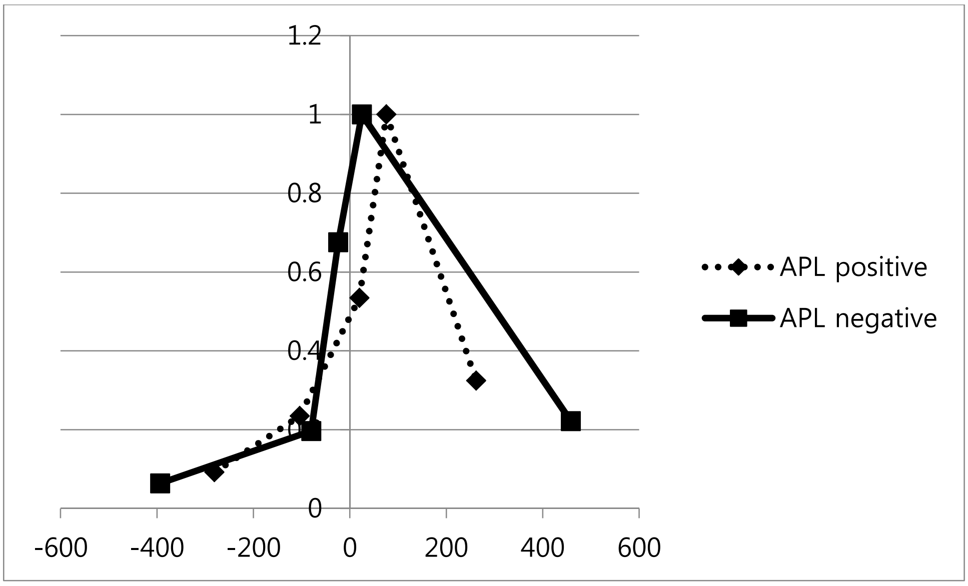

Figure 8. FCR average sEMG amplitude normalized to peak plotted against time in milliseconds. Impact is plotted at O seconds. Note the negative response occurring later from higher amplitude with later peak.

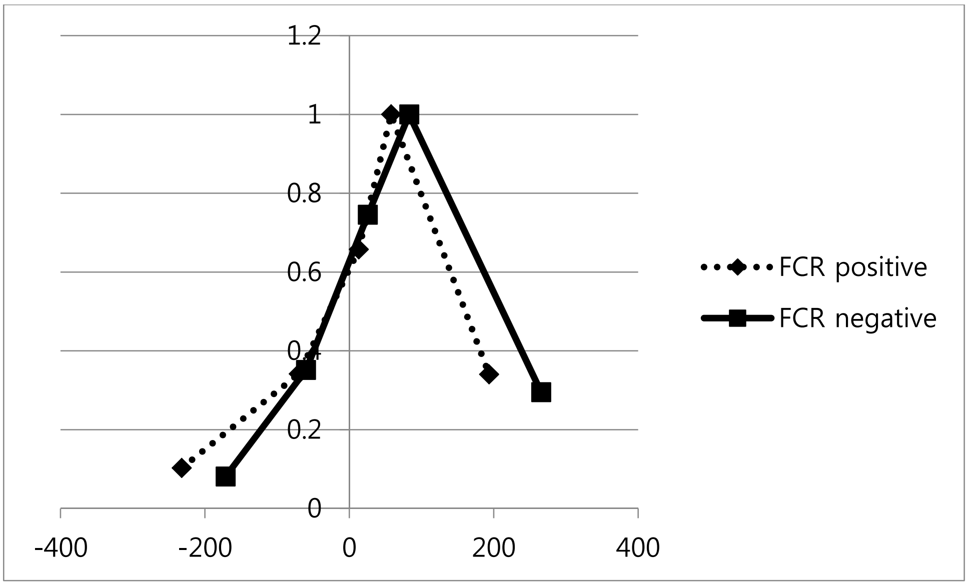

Figure 9.

FCU average sEMG amplitude normalized to peak plotted against time in milliseconds. Impact is plotted at O seconds. Note the negative response occurring earlier from higher amplitude.